

OBESITY AND THYROID DYSFUNCTION: HORMONAL MECHANISMS AND MODERN TREATMENT STRATEGIES

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Abstract. Obesity and thyroid dysfunction are closely related endocrine disorders. Thyroid hormones regulate energy expenditure and metabolism, while excess adipose tissue may influence thyroid function through hormonal and inflammatory mechanisms. Hypothyroidism may contribute to weight gain and dyslipidemia, whereas obesity can cause changes in thyroid function tests. Early diagnosis and individualized treatment are important for improving metabolic health and preventing complications.

Keywords: Obesity; thyroid dysfunction; hypothyroidism; thyroid hormones; metabolism; TSH; body weight.

Introduction

Obesity and thyroid dysfunction are two major endocrine and metabolic conditions that have become increasingly important public health concerns worldwide. The prevalence of obesity has increased substantially in recent decades, contributing to a growing burden of metabolic, cardiovascular, and endocrine disorders. At the same time, thyroid dysfunction, particularly hypothyroidism, is frequently identified in individuals with increased body weight.

The coexistence of obesity and thyroid abnormalities has attracted considerable clinical interest because of the complex relationship between energy metabolism, hormonal regulation, and body weight. Thyroid hormones, mainly thyroxine (T4) and triiodothyronine (T3), play a central role in regulating basal metabolic rate, thermogenesis, oxygen consumption, and energy expenditure.

Alterations in thyroid hormone availability may therefore influence body weight and adipose tissue metabolism. Reduced thyroid hormone activity can decrease energy expenditure and may contribute to weight gain, although the extent of this effect varies among individuals.

Conversely, increased adiposity may itself influence thyroid function through changes in hormone metabolism, deiodinase activity, and hypothalamic-pituitary-thyroid axis regulation.

Relevance

Obesity and thyroid dysfunction are common endocrine disorders with a close metabolic relationship. Thyroid hormones regulate energy expenditure, lipid metabolism, and body weight, while excess adipose tissue may alter thyroid function through hormonal and inflammatory mechanisms.

Main part

Thyroid hormones, particularly triiodothyronine (T3) and thyroxine (T4), play a central role in the regulation of energy expenditure, thermogenesis, lipid metabolism, and overall metabolic homeostasis. Reduced thyroid hormone activity may decrease basal metabolic rate and energy expenditure, potentially contributing to weight gain. However, the relationship between obesity and thyroid dysfunction is bidirectional and considerably more complex.

Adipose tissue is an active endocrine organ that produces leptin, adipokines, and inflammatory mediators capable of influencing the hypothalamic-pituitary-thyroid axis. Increased leptin concentrations in obesity may affect hypothalamic regulation of thyrotropin secretion and consequently alter thyroid hormone metabolism. Obesity may also influence the peripheral conversion of T4 into T3 through changes in deiodinase activity. In addition, insulin resistance and chronic low-grade inflammation associated with excessive adiposity may contribute to disturbances in endocrine regulation. These mechanisms can result in mild alterations in thyroid function tests even in the absence of clinically significant thyroid disease. At the same time, established hypothyroidism may contribute to dyslipidemia, reduced energy expenditure, and difficulties in controlling body weight. Therefore, obesity should not be considered simply a consequence of thyroid dysfunction. The interaction involves multiple hormonal, metabolic, and inflammatory pathways that influence both energy balance and thyroid function.

The diagnosis of thyroid dysfunction in patients with obesity can be challenging because several clinical manifestations overlap between the two conditions. Weight gain, fatigue, reduced physical activity, and changes in metabolic parameters may occur in both obesity and hypothyroidism. For this reason, clinical symptoms alone are insufficient to establish thyroid dysfunction. Laboratory assessment plays a central role in diagnosis, with thyroid-stimulating hormone (TSH) generally serving as the primary screening marker. Measurement of free thyroxine (FT4) provides additional information and helps distinguish different forms of thyroid dysfunction.

In selected patients, thyroid autoantibodies may be assessed when autoimmune thyroid disease is suspected. It is important to interpret thyroid function tests within the clinical context because mild TSH elevation may occur in individuals with obesity without overt hypothyroidism.

Consequently, an isolated abnormal laboratory value should not automatically be considered evidence that thyroid dysfunction is the primary cause of excessive body weight. A comprehensive evaluation should also include body mass index, waist circumference, blood pressure, fasting glucose, and lipid profile. When structural thyroid abnormalities are suspected, ultrasonography may provide additional diagnostic information. Appropriate diagnosis is essential because unnecessary thyroid hormone treatment in patients without confirmed thyroid disease may provide limited benefit and expose patients to potential adverse effects. Conversely, failure to identify clinically relevant hypothyroidism may contribute to persistent metabolic abnormalities.

The management of patients with obesity and thyroid dysfunction should be individualized according to the underlying endocrine condition, degree of obesity, metabolic risk factors, and associated complications. In patients with confirmed hypothyroidism, appropriate thyroid hormone replacement remains the standard treatment, with the objective of restoring adequate thyroid function and reducing the clinical consequences of hormone deficiency. However, correction of hypothyroidism alone does not usually produce substantial weight loss when obesity has multiple metabolic and behavioral causes. Therefore, obesity should be addressed through a comprehensive strategy that includes dietary modification, regular physical activity, behavioral interventions, and appropriate long-term monitoring. For patients who meet established clinical criteria, pharmacological treatment may be considered to support clinically meaningful weight reduction.

Modern anti-obesity therapies can influence appetite regulation, food intake, and metabolic pathways and should be selected according to individual characteristics and contraindications. In patients with severe obesity and significant obesity-related complications, bariatric surgery may represent an effective therapeutic option. Thyroid function should continue to be monitored during significant changes in body weight because metabolic changes may influence hormone requirements in some patients. Management should also include assessment of glucose metabolism, lipid profile, blood pressure, and cardiovascular risk. An integrated approach that simultaneously addresses thyroid function and obesity-related metabolic abnormalities may improve overall health outcomes and reduce the risk of long-term complications.

Conclusion

Obesity and thyroid dysfunction have a complex relationship involving hormonal, metabolic, and inflammatory mechanisms. Thyroid hormones influence energy expenditure and body weight, while excessive adipose tissue can affect thyroid function. Accurate diagnosis is therefore essential to distinguish clinically significant thyroid disease from obesity-related changes in thyroid parameters. Effective management should combine appropriate treatment of thyroid dysfunction with lifestyle modification and evidence-based obesity therapy. An individualized and comprehensive approach can improve metabolic health and reduce the risk of long-term complications.

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